Pulse Oximetry: Uses and Limitations

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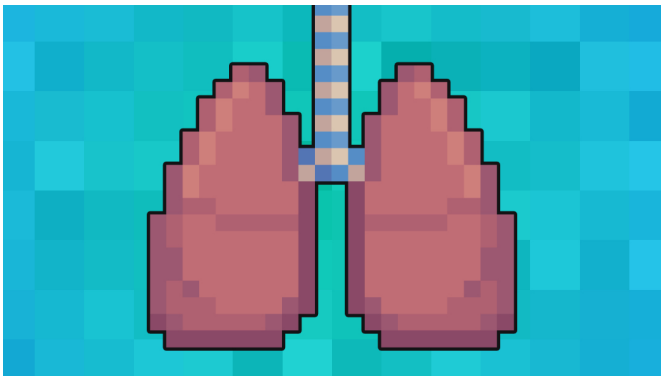

You will see **pulse oximetry** being used in a number of clinical settings, such as in the emergency department and in the operating theatre, to assess and monitor patients, so it is important to know about its uses and limitations, as well as how to interpret the readings that it produces.

The fundamental principle behind pulse oximetry is that when you shine light of a certain wavelength at molecules of oxygenated and deoxygenated haemoglobin differing amounts of light are absorbed by these molecules. So, if you place a light source emitting these specific wavelengths of light on one side of the finger and a sensor that detects these wavelengths of light on the other side, one can measure the amount of light being absorbed within the tissue by oxygenated and deoxygenated haemoglobin. The reading that is produced (the SpO_2) represents the percentage of oxygenated haemoglobin present as a proportion of the total amount of haemoglobin detected. So, a reading of 92% means that the pulse oximeter has detected that 92% of the haemoglobin molecules sampled are carrying oxygen and 8% are deoxygenated molecules. Pulse oximeters are designed to provide readings on haemoglobin molecules that are travelling in a pulsatile manner, so the reading represents the situation that exists in the arterial circulation. You will see pulse oximetry probes being applied to fingers and toes and also to hands, feet and earlobes in babies and you may encounter reflectance probes that take readings from a flat body surface by reflecting light off the skull, or sternum.

Knowing how a pulse oximeter measures the SpO_2 is important because it helps you to understand what its limitations are and why you can experience erroneous readings, or no readings at all. Like any other item of equipment used to assess and monitor patients, it is not perfect and it can produce incorrect readings. The two most commonly encountered problems are as follows:

Light contamination/interference

Pulse oximeters depend upon the sensor being able to detect light being shone through body tissue and to measure the amount of light being absorbed by oxygenated haemoglobin. If ambient light

enters the probe site, this can cause the pulse oximeter not to produce a reading at all, or to produce an erroneous reading. Many pulse oximetry finger probes are designed to shield the finger from ambient light but this can be negated if a small finger clip probe is applied to a digit that is too large, such as a big toe. Equally, if a finger clip probe that is too large is applied to a digit that is too small, there will not be any finger tissue between the light source located in one arm of the probe and the sensor that is located in the opposite arm. Another problem that may be encountered is painted fingernails, which may either reflect or absorb light being shone at them, depending upon the colour of the varnish.

Pulsatile haemoglobin

Pulse oximeters are designed to detect haemoglobin molecules that are moving in a pulsatile fashion. In situations where the patient's peripheral circulation is sluggish, such as in peripheral shutdown due to shock, or local hypothermia, the pulse oximeter may not be able to detect pulsatile movement. This may result in either no readings, or erroneous readings being produced. Similarly, if the patient is shivering, has a tremor, or is moving the finger excessively, the ability to provide an accurate reading may also be affected. (This is why it is important in medicine to always check the patient, as well as the machine!) Pulse oximeters now have sophisticated software that is designed to detect movement artefact and low output states within the tissue being sampled, as well as visual and audible methods of assessing the quality of the signal being received (bar graph & plethysmographic representations of signal strength and quality), so it is important to use these systems to assess the quality of the information that is being delivered by the pulse oximeter. (It is good practice to assess the patient's pulse at the wrist and compare it to the pulse rate reading produced by the pulse oximeter, as well as to check a capillary refill time before applying the probe to the finger.)

Oxygen saturation readings

For a cell to survive and function efficiently, it requires a good supply of oxygen so that aerobic metabolism can take place. For this to occur, oxygen needs to be uploaded onto the haemoglobin carrier molecules, which takes place at the alveolar interface between the respiratory and circulatory systems and then downloaded within the individual organs. The oxygen saturation reading (SpO_2) is a measurement of the amount of oxygenated haemoglobin being carried within the circulatory system. Therefore, it does not provide a full picture of how well a patient is ventilating, nor does it provide information on the cellular environment that can impact on the ability of the haemoglobin molecule to download oxygen and for the cellular uptake of oxygen. This is why a blood gas measurement is required, as information on the CO_2 level, pH balance, etc is required to provide a complete picture of the patient's respiratory function and cellular function. For example, in cyanide poisoning, the SpO_2 is normal but the cells are hypoxic because the poison prevents the downloading of oxygen from haemoglobin to the cells. Thus, pulse oximetry is really providing information on how well oxygen is being uploaded to the haemoglobin molecules and it is inferred from this reading that the patient is either hypoxic or not, although cellular hypoxia may still be

present.

Clinical application of pulse oximetry

There are a number of factors that can adversely affect the uploading of oxygen at the alveolar interface between the respiratory and circulatory systems and it is helpful to think of the alveolar membrane as having a respiratory surface and a circulatory surface in considering these. The causes of a low SpO_2 are listed at the end of this article, but understanding why these are causes is also helpful. Follow the oxygen molecule from the atmosphere to the bloodstream and then consider the factors that would prevent oxygen from coming into contact with haemoglobin.

The first factor to be considered is the amount of oxygen present in the atmosphere available to be inhaled, which varies with altitude and the presence of other gases replacing oxygen in the atmosphere, such as carbon monoxide. The volume of oxygen that is inhaled is a function of both the respiratory rate and the respiratory depth, so anything that causes respiratory depression (head injuries, respiratory depressant drugs) will reduce the respiratory rate and also the respiratory depth. A number of other conditions will also reduce the respiratory depth, such as restricted chest expansion from splinting of the chest wall due to pain from rib fractures. Any condition that interferes with the ability to create negative intrathoracic pressure within the chest, which is required to draw air down the trachea, will result in less oxygen entering the lungs. This is the mechanism by which patients with a flail chest, or tension pneumothorax; with muscular or neurological conditions affecting the ability of the chest to expand; or with conditions affecting the excursion of the diaphragm, become hypoxic. Upper and lower airway obstruction from aspiration of the tongue, food or a foreign body, as well as from infection, or bronchospasm will reduce the delivery of oxygen to the alveoli. Within the alveoli, the respiratory side of the interface can be obstructed by fluid such as pulmonary oedema, pus and airway secretions. On the circulatory side of the interface, anything that decreases the delivery of haemoglobin to the alveoli will prevent the uploading of oxygen, as is the case with a pulmonary embolus, or in decreased blood flow due to profound shock.

Thus, when one is presented with a patient with a low SpO_2, it is important to consider all the potential causes for this, especially if a clear cause is not immediately obvious, or if the patient does not respond to treatment.

Pulse oximetry as an assessment tool

Pulse oximetry has been used to assess and monitor acutely unwell patients for many years and measuring the SpO_2 is now almost routine in many clinical settings, along with the other vital signs. Many guidelines now use the SpO_2 reading to determine whether or not action needs to be taken, or oxygen therapy initiated. It is important to recognise that the oxyhaemoglobin dissociation curve has a very steep curve to it, such that small drops in the SpO_2 may have great physiological and clinical significance. This simply reflects the importance of O_2 in aerobic cellular metabolism and the way

that the body's compensatory mechanisms function to try and maintain a 100% oxygen saturation level. In previously fit patients, with normal respiratory and circulatory systems, a fall in the SpO_2 of around 5-6% from normal should indicate the need for a careful assessment of the patient's other physiological parameters, such as the heart and respiratory rates, to establish how well they are compensating for the low SpO_2. An holistic clinical assessment of the patient and of these physiological parameters is required. However, a fall in the SpO_2 of 8-10% represents a significant degree of hypoxia and should trigger an immediate and thorough clinical assessment to establish the cause, along with the initiation of oxygen therapy and disease-specific treatment, especially in the presence of tachycardia and tachypnoea.

The potential for sudden and unexpected decompensation in these patients is significant and they are likely to be very symptomatic, in terms of shortness of breath and apprehension, progressing to agitation, confusion, restlessness and then a rapidly increasing level of unresponsiveness. As hypoxia progresses further, the patient may become bradycardic and the respiratory rate may fall, as hypoxia-induced respiratory depression ensues. It should be noted, though, that patients with COPD develop compensatory mechanisms for coping with a chronically low SpO_2, such as increasing the number of haemoglobin molecules available for oxygen uploading, or switching to the hypoxic drive mechanism. SpO_2 readings in these patients need to be seen within the context of other physiological parameters, such as the mental state, pulse and respiratory rates and blood gases, in order to identify those patients in respiratory failure.

Pulse oximetry as a screening tool

Whilst pulse oximetry can be of great value in assessing patients with either known respiratory conditions, or with symptoms suggestive of respiratory conditions, such as breathlessness, cough or noisy breathing, it can also be of value as a screening tool to detect serious medical conditions that adversely affect oxygen uploading at the alveolar interface. Two such conditions are an atypical pneumonia and pulmonary embolus syndrome (PES), where respiratory symptoms may not be the predominant ones, or may be absent. Here, the presence of a low SpO_2 may be the only indicator that one is dealing with these two potentially life-threatening conditions. There is one caveat to this, however, which is that the SpO_2 may need to be assessed after exertion and not just at rest to expose the ventilation/perfusion deficit, especially if the patient is complaining of shortness of breath on exertion, rather than breathlessness at rest.

The following two cases highlight the benefits of taking this approach

Case 1

Mr Jones is a 54 year old male who reports that he has had a "flu-like" illness for 2 days with symptoms of "*feeling hot and aching all over*". Symptom-based questioning provided no clues as to the focus of the infection, although a risk assessment revealed that he had been to a spa hotel during

the previous week. He was normally fit and well and had no previous medical history of note. His observations showed a regular pulse of 110, BP of 130/90, respiratory rate of 20 and a temperature of 38.6°c. His SpO_2 was 92%, however. Clinical examination of his chest was unremarkable. He denied having any respiratory symptoms, in terms of a cough, chest pain, or dyspnoea.

The presence of a fever with a low SpO_2 should always raise the possibility that the patient has an atypical pneumonia, regardless of the absence of respiratory symptoms and positive findings in the chest examination. The history of a spa holiday raises the question of hot tub usage, which is associated with the potential for Legionnaire's disease. He was referred to the Emergency Assessment Unit and a CXR revealed an area of consolidation, with Legionnaire's antigen testing positive in his urine. His liver function tests were also noted to be grossly deranged. Within hours of arriving in hospital, the patient became increasingly unwell from multi-organ failure, requiring transfer to the intensive care unit. With intravenous antibiotics and supportive care, the patient made a full recovery. All patients with a pyrexia of unknown origin should have pulse oximetry performed, as this may indicate the presence of an atypical pneumonia.

Case 2

Jenny is a 24 year old woman with a 3-week history of increasing shortness of breath on exertion. Today she was forced to stop and rest several times whilst walking the dog. On examination, Jenny was sitting comfortably and talking easily, without interruption to draw breath. At rest, her observations revealed a regular pulse rate of 100 bpm, BP of 120/80, respiratory rate of 22 and a normal temperature. Her SpO_2 was 96%. Examination of her chest was unremarkable. As her symptom was of shortness of breath on exertion, she was asked to walk around the room a few times and was then re-assessed. Her heart rate had increased to 132 bpm, whilst her respiratory rate increased to 38. The SpO_2 fell to 84% and Jenny became quite breathless and uncomfortable, although her symptoms eased rapidly with rest and 100% oxygen. Her SpO_2 remained stubbornly low at 95%, despite oxygen therapy.

Jenny's presentation is consistent with a diagnosis of pulmonary embolism, in that the deterioration in her condition had not been gradual but stepwise, which indicates that a series of events has taken place, as is experienced in patients with multiple pulmonary emboli. Her Wells Score was negative and there were no identifiable risk factors. Jenny was referred to the assessment unit for a D-Dimer and CT-PA, both of which were positive, with multiple pulmonary emboli in both lung fields being diagnosed.

It is important to recognise both the typical and atypical presentations of this serious illness and a recent European study in to the symptoms and signs of pulmonary embolus has highlighted the need to review our knowledge of how patients with a PE present. This is because we need to be able to use this information when we match the patient's symptoms and signs to a pattern of illness during the clinical reasoning process and also to identify the red flags that might be present. The study found that only 50% of patients with a PE present with dyspnoea; 39% with pleuritic chest pain; 15%

with substernal chest pain; and only 24% had clinical signs of a lower limb venous thromboembolism (VTE). In particular, patients with large, or centrally-located PE often present with non-specific symptoms and signs, such as unexplained syncope or hypotension, sweating, central chest pain, or persistent dyspnoea. Of greater concern though, was the finding that over 40% of patients who died from a PE were undiagnosed ante-mortem and that only 7% of the patients who died early were correctly diagnosed as having had a pulmonary embolus before death.

The reason that pulse oximetry is of value is that over 60% of patients with a PE will have a low SpO_2, although it may be necessary to exert the patient to expose the ventilation/perfusion deficit, especially if the symptom is breathlessness on exertion, rather than at rest. As cyanosis may not become clinically apparent until the SpO_2 falls below 85%, pulse oximetry is an important tool for detecting hypoxia, as other parameters, such as the heart rate and respiratory rate may be affected by factors other than hypoxia, such as hypovolaemia, fever and anxiety. These two cases demonstrate the importance of using pulse oximetry as a screening tool in the clinical assessment of all acutely unwell patients to detect the presence of hypoxia and what may be atypical presentations of life-threatening acute illness.

In summary

Pulse oximetry is not a perfect tool and has both clinical and practical limitations to its use. The SpO_2 reading must always be considered within the context of the rest of the patient's clinical assessment. However, there is an old saying in medicine, which is this: "If you don't take a temperature, you won't find a fever". The same applies to pulse oximetry, "if you don't measure the SpO_2, you won't find hypoxia". Illness is a dynamic process and the earlier that ones sees a patient, the more difficult it can be to establish the diagnosis, so any physiological measurement that can alert us to the presence of serious illness, or that help to quantify the severity of that illness, is valuable in clinical assessment.

Key points

Pulse oximetry measures the amount of oxygenated haemoglobin circulating in the peripheral arterial circulation, presenting this as a percentage of the total haemoglobin present.

A low SpO_2 indicates that either oxygen is not reaching the alveoli, or it is not being uploaded onto haemoglobin molecules and transported to the peripheral arterial circulation.

Pulse oximetry is not a perfect tool and its readings can be adversely affected by a number of clinical and environmental factors.

Pulse oximetry can be used to assess and monitor patients with known respiratory conditions, or with respiratory symptoms to determine the severity of the illness and also as a screening tool in acutely unwell patients to detect conditions that can give rise to hypoxia.

Pulse oximetry can be used to decide how to manage a patient, in terms of whether or not oxygen therapy is required and whether the patient should be admitted to hospital or not. SpO_2 levels feature in many of the risk assessment and clinical decision-making tools in various NICE/BTS/SIGN guidelines.

Causes of a low SpO_2

- Reduced atmospheric oxygen (e.g. high altitude, carbon monoxide-rich environment)
- Respiratory depression (e.g. head injury, drug overdose)
- Upper airway obstruction (e.g. epiglottitis, croup, inhaled foreign body)
- Lower airway obstruction (e.g. bronchospasm, lung collapse/consolidation)
- Restriction to chest/diaphragmatic expansion (e.g. pain from rib fractures, neurological conditions)
- Restriction to lung expansion (haemothorax, large pleural effusion)
- Inability to create negative intrathoracic pressure (flail chest, tension pneumothorax)
- Obstruction to oxygen transfer across the alveolar wall (e.g. pulmonary oedema)

References

The World Health Organisation (WHO) has produced two excellent short tutorials on the principles and use of pulse oximetry in patient assessment, which can be viewed on-line. (http://www.who.int/patientsafety/safesurgery/pulse_oximetry/tr_material/en/)

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